

A longitudinal analysis of women's salivary testosterone and intrasexual competitiveness

Short title: Sex hormones and intrasexual competition

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Abstract

Research on within-subject changes in women's intrasexual competitiveness has generally focused on possible relationships between women's intrasexual competitiveness and estimates of their fertility. While this approach is useful for testing hypotheses about the adaptive function of changes in women's intrasexual competitiveness, it offers little insight into the proximate mechanisms through which such changes might occur. To investigate this issue, we carried out a longitudinal study of the hormonal correlates of changes in intrasexual competitiveness in a large sample of heterosexual women (N=136). Each woman provided saliva samples and completed an intrasexual competitiveness questionnaire in five weekly test sessions. Multilevel modeling of these data revealed a significant, positive within-subject effect of testosterone on intrasexual competitiveness, indicating that women reported greater intrasexual competitiveness when testosterone was high. By contrast, there were no significant effects of estradiol, progesterone, estradiol-to-progesterone ratio, or cortisol and no significant effects of any hormones on reported relationship jealousy. This is the first study to demonstrate correlated changes in measured testosterone levels and women's reported intrasexual competitiveness, implicating testosterone in the regulation of women's intrasexual competitiveness.

Keywords: intrasexual competition, sex hormones, stress, jealousy, within-sex competition, relationships

1. Introduction

Intrasexual competition refers to competition between individuals of the same sex for access to mating opportunities (Andersson, 1994). Most work on intrasexual competition in humans has focused on direct (i.e., physically aggressive) competition among men (Stockley & Campbell, 2013). Although direct competition among women clearly does occur (Stockley & Campbell, 2013), intrasexual competition among women more commonly takes other forms, such as self-promotion and the derogation of competitors (Vaillancourt, 2013).

Studies investigating the physiological factors that may be implicated in changes in women's intrasexual competitiveness have generally focused on a possible relationship between estimates of women's fertility and measures of their intrasexual competitiveness. For example, during the high-fertility phase of their menstrual cycle, women are more motivated to dress sexily (Durante et al., 2008; Haselton et al., 2007), more likely to purchase desirable consumer goods (Durante et al., 2011), and more likely to withhold resources from attractive women (Lucas & Koff, 2013). While these results link women's fertility to the extent to which they engage in self-promotion, other work has examined the relationship between fertility and the extent to which women derogate other women's attractiveness. For example, young women are more likely to derogate other women's attractiveness during the high-fertility phase of their menstrual cycle than during other phases (Fisher, 2004). Similarly, pre-menopausal women are more likely to derogate other women's attractiveness than are post-menopausal women (Vukovic et al., 2009). Increases in intrasexual competitiveness around ovulation, such as those described above, are suggested to occur because successful competition for mating opportunities with high-quality mates is more likely to translate into fitness benefits at this time (Durante et al., 2008, 2011; Fisher, 2004; Haselton et al., 2007; Lucas & Koff, 2013; Vukovic et al., 2009).

The approach adopted by the studies described in the previous paragraph (i.e., linking changes in competitiveness to fertility) is useful for testing hypotheses about the adaptive

function of changes in women's intrasexual competitiveness. However, it offers little insight into the proximate mechanisms through which such changes might occur. In an effort to address this issue, recent work has compared women's intrasexual competitiveness during the late-follicular and mid-luteal cycle phases and following hormonal contraceptive use in an effort to infer the hormonal correlates of changes in women's intrasexual competitiveness (Cobey et al., 2013). Cobey et al. (2013) found that partnered women's reported intrasexual competitiveness was lower following hormonal contraceptive use than it was during either the late-follicular or mid-luteal phases of the cycle when these women were not using hormonal contraceptives. Moreover, intrasexual competitiveness did not differ between these late-follicular or mid-luteal phases. Since hormonal contraceptive use lowers testosterone levels in women (Zimmerman et al., 2014) and evidence that testosterone levels change between the late-follicular and mid-luteal phases is mixed (Dabbs, 1990; Dabbs & de La Rue, 1991; see also Caruso et al., 2014), Cobey et al. (2013) speculated that the observed changes in women's intrasexual competitiveness may be a consequence of changes in their testosterone levels. Indeed, this explanation would be consistent with findings showing that women's testosterone levels increased after they imagined their partner flirting with an attractive woman (Ritchie & van Anders, 2014) or were exposed to olfactory cues associated with ovulation in other women (Maner & McNulty, 2013). This explanation would also be consistent with findings from research with some non-human animals (e.g., birds and rats), which suggests that testosterone administration increases intrasexual competitiveness (Albert et al., 1990; Zysling et al., 2006).

Although Cobey et al. (2013) suggested that women's intrasexual competitiveness may track naturally occurring changes in their testosterone levels, no previous studies have tested for correlated changes in women's intrasexual competitiveness and measured testosterone levels. Thus, we investigated the hormonal correlates of within-subject changes in women's reported intrasexual competitiveness. We did this using a longitudinal design, in which women reported their intrasexual competitiveness and provided saliva samples in five

consecutive weekly test sessions. Following Cobey et al. (2013), we assessed intrasexual competitiveness using Buunk and Fisher's (2009) intrasexual competitiveness scale. Our analyses considered the possible effects of testosterone, estradiol, progesterone, estradiol-to-progesterone ratio, and cortisol, as well as women's partnership status. This type of design has recently been used to investigate the hormonal correlates of changes in women's responses to facial and vocal cues (Hahn et al., 2015; Pisanki et al., 2014; Wang et al., 2014) and appearance (Jones et al., 2015).

While some research implicates testosterone in the regulation of women's intrasexual competitiveness (Cobey et al., 2013; Maner & McNulty, 2013; Ritchie & van Anders, 2014), other work suggests that relationship jealousy (i.e., the extent to which women would become jealous at the thought or observation of their partner interacting with another woman) varies as a function of women's estradiol. Geary et al. (2001) reported that salivary estradiol and reported jealousy were positively correlated and Cobey et al. (2012) found that women reported greater jealousy during the high-fertility, late-follicular phase of their menstrual cycle and that partnered women reported greater jealousy following hormonal contraceptive use. Reported jealousy (Cobey et al., 2011) is also greater among women using high-estrogen hormonal contraceptives than among women using low-estrogen hormonal contraceptives. In light of these findings, we also investigated the hormonal correlates of changes in women's reported relationship jealousy. We assessed relationship jealousy using Buunk's (1997) jealousy scale, which has previously been used in work investigating differences in jealousy as a function of hormonal contraceptive use and cycle phase (Cobey et al., 2012) and hormonal contraceptive estrogen dosage (Cobey et al., 2011). That previous research has linked women's reported relationship jealousy and intrasexual competitiveness to estradiol and testosterone respectively, suggests that reported relationship jealousy and intrasexual competitiveness may be related, but dissociable, behaviors.

2. Methods

2.1 Participants

Participants were 141 heterosexual women (mean age=21.58 years, SD=3.12 years) at the University of Glasgow. Participants were recruited via an advert circulated to all women registered with the School of Psychology (University of Glasgow) participant pool.

Participants were recruited only if they were not currently using any hormonal supplements (e.g., oral contraceptives) and had not used any form of hormonal supplements in the 90 days prior to their participation. None of the participants reported being pregnant, having been pregnant recently, or breastfeeding. Forty-seven of the women reported that they were currently in a romantic relationship and 94 of the women reported that they were not. Each participant completed five consecutive weekly test sessions. Data on 45 of these women's voice preferences are reported in Pisanski et al. (2014). Data on the reward value of adult facial attractiveness and infant facial cuteness for 39 and 45 of these women are reported in Wang et al. (2014) and Hahn et al. (2015), respectively. Data on the facial coloration of 64 of these women are reported in Jones et al. (2015). Data on 44 of these women's makeup preferences are reported in Fisher et al. (under review). Note that, other than the hormone values, there was no overlap in the data analyzed across these pieces of work.

2.2 Assessing intrasexual competitiveness and relationship jealousy

In each test session, participants completed Buunk and Fisher's (2009) intrasexual competitiveness scale and Buunk's (1997) jealousy scale, following Cobey et al. (2013) and Cobey et al. (2012), respectively. Buunk and Fisher's (2009) intrasexual competitiveness scale is a 12-item questionnaire on which participants indicate how applicable each item is to them using a one to seven scale, with higher scores indicating greater intrasexual competitiveness. Examples of scale items include, "I want to be just a little better than other women" and "I tend to look for negative characteristics in women who are very successful". Following (Buunk & Fisher, 2009), scores for the 12 items were averaged; the mean score for the sample was 2.76 (SD=1.09). Consistency across items was high (Cronbach's

alpha=.90). Previous research has demonstrated that responses on this scale are sensitive to contextual factors (Buunk & Massar, 2012; see also Cobey et al., 2013), suggesting it is appropriate for detecting changes in reported intrasexual competitiveness.

Buunk's (1997) jealousy scale is a 15-item questionnaire on which answers are reported on a one to five scale, with higher scores indicating higher levels of jealousy. Examples of scale items include "I am concerned that my partner finds someone else more attractive than me", "It is unacceptable to me that my partner has friends of the opposite sex", and "How would you feel if your partner would dance intimately with someone of the opposite sex?".

Following Cobey et al. (2011; 2012), partnered women were instructed to consider these questions in the context of their current romantic partner and unpartnered women were instructed to consider these items in the context of their last romantic partner. Following (Cobey et al., 2012), scores for the 15 items were summed; the mean score for the sample was 38.39 (SD=9.52). Consistency across items was high (Cronbach's alpha=.88).

2.3 Hormone assays

Participants provided a saliva sample via passive drool (Papacosta & Nassis, 2011) in each test session. Participants were instructed to avoid consuming alcohol and coffee in the 12 hours prior to participation and avoid eating, smoking, drinking, chewing gum, or brushing their teeth in the 60 minutes prior to participation. Each woman's test sessions took place at approximately the same time of day to control for possible effects of diurnal changes in hormone levels (Veldhuis et al., 1988; Bao et al., 2003).

Saliva samples were frozen immediately and stored at -32°C until being shipped, on dry ice, to the Salimetrics Lab (Suffolk, UK) for analysis, where they were assayed using the Salivary 17 β -Estradiol Enzyme Immunoassay Kit 1-3702 (M=3.96 pg/mL, SD=1.16 pg/mL, sensitivity=0.1 pg/mL, intra-assay CV=7.13%, inter-assay CV=7.45%), Salivary

Progesterone Enzyme Immunoassay Kit 1-1502 (M=149.66 pg/mL, SD=66.84 pg/mL, sensitivity=5 pg/mL, intra-assay CV=6.20%, inter-assay CV=7.55%), Salivary Testosterone Enzyme Immunoassay Kit 1-2402 (M=85.96 pg/mL, SD=20.72 pg/mL, sensitivity<1.0 pg/mL, intra-assay CV=4.60%, inter-assay CV=9.83%), Salivary Cortisol Enzyme Immunoassay Kit 1-3002 (M=0.30 µg/dL, SD=0.62 µg/dL, sensitivity<0.003 µg/dL, intra-assay CV=3.50%, inter-assay CV=5.08%). All assays passed Salimetrics' quality control. We also calculated estradiol-to-progesterone ratio for each woman's individual test sessions (M=0.04, SD=0.04). Five women were removed from the dataset at this point because they had at least one test session with an atypically high or low progesterone level (N=2) or cortisol level (N=3). We note here, however, that the pattern of significant results described in our main analyses was the same when these five women were retained in the dataset.

2.4 Analyses

We tested for within-subject effects of salivary estradiol, progesterone, testosterone, estradiol-to-progesterone ratio, and cortisol on reported intrasexual competitiveness and relationship jealousy using multilevel modeling with test sessions grouped by participant (five test sessions per participant). Analyses were conducted using R (R Core Team, 2013), *lme4* (Bates et al., 2014), and *lmerTest* (Kuznetsova et al., 2013). To test for within-subject effects of hormone levels on reported intrasexual competitiveness and jealousy, scores on the intrasexual competitiveness and jealousy scales were entered as the dependent variable at the test session level and values for salivary estradiol, progesterone, testosterone, estradiol-to-progesterone ratio, and cortisol were simultaneously entered as predictors, again at the test session level. Estradiol-to-progesterone level was included in our analyses in addition to estradiol and progesterone because it is positively correlated with fertility in women not using hormonal contraceptives (Baird et al., 1991; Landgren et al., 1980). Cortisol was included in our analyses in addition to testosterone because of previous work demonstrating that engaging in intrasexual competition increases women's cortisol and testosterone levels (Bateup et al., 2002; Casto & Edwards, in press). All continuous predictors were centered on

their grand means (following, e.g., Puts et al., 2013) and women's partnership status was included as a between-subject factor (0 = unpartnered, 1 = partnered) entered at the participant level. Initial models included interactions between partnership status and each hormone, in addition to the effects of individual hormones and partnership status. Changes in intrasexual competitiveness and jealousy were investigated in separate analyses. The full outputs for each of our main analyses are included in our supplemental materials. Our supplemental materials also include a table reporting descriptive statistics for each participant's maximal and minimal values for each variable.

3. Results

All analyses reported below include data from 136 women (see 2.3 Hormone assays for details of five excluded participants).

3.1 *Intrasexual competitiveness*

Since our initial model for scores on the intrasexual competitiveness scale showed no interactions between partnership status and any of the hormones (all $|t| < 1.08$, all $p > .28$), these interactions were removed from the model. This reduced model revealed a significant, positive within-subject effect of testosterone ($t = 2.05$, unstandardized beta = 0.003, $p = .041$, Figure 1), indicating that women reported stronger feelings of intrasexual competitiveness in test sessions where testosterone was high. There were no other significant within-subject effects (all $|t| < 1.55$, all $p > .12$) and the between-subject effect of partnership status was also not significant ($t = -0.69$, unstandardized beta = -0.135 , $p = .49$). This pattern of results was not altered when estradiol-to-progesterone ratio was removed from the model and estradiol, progesterone, testosterone, and cortisol were retained or when estradiol and progesterone were removed from the model and estradiol-to-progesterone ratio, testosterone, and cortisol were retained. A model including testosterone as the only predictor also showed a significant, positive within-subject effect of testosterone ($t = 2.50$, unstandardized beta = 0.003, $p = .013$). This effect of testosterone was marginally significant when this analysis was repeated with test session order included as an additional

predictor ($t = 1.90$, unstandardized beta = 0.001, $p = .058$), suggesting the tendency for intrasexual competitiveness to be greater when testosterone levels are high is not simply an artifact of the effects of test session order. This analysis also showed a significant negative effect of test session order ($t = -6.32$, unstandardized beta = -0.067 , $p < .001$).

INSERT FIGURE 1 HERE

Since previous work suggests that engaging in intrasexual competition increases women's testosterone and cortisol (Bateup et al., 2002; Casto & Edwards, in press), we also ran an additional model with cortisol as the only predictor. This model did not show a significant within-subject effect of cortisol ($t = 0.73$, unstandardized beta = 0.108, $p = .47$). Repeating these analyses controlling for the possible effects of participant age on intrasexual competitiveness showed the same pattern of significant results in all cases and none of these analyses showed significant effects of participant age.

Repeating each of our analyses of reported intrasexual competitiveness, this time with continuous predictors centered on subject-specific, rather than grand, means showed the same pattern of significant results (see our supplemental materials for full outputs).

3.2 Relationship jealousy

Our initial model for scores on the jealousy scale also showed no interactions between partnership status and any of the hormones (all $|t| < 1.30$, all $p > .19$), so these interactions were removed from the model. This reduced model revealed a significant between-subject effect of partnership status ($t = -2.14$, unstandardized beta = -3.630 , $p = .034$), indicating that partnered women generally reported less jealousy ($M=35.97$) than did unpartnered women ($M=39.64$). There were no significant within-subject effects of any of the hormones (all $|t| < 1.51$, all $p > .13$). This pattern of results was not altered when estradiol-to-progesterone ratio was removed from the model and estradiol, progesterone, testosterone,

and cortisol were retained or when estradiol and progesterone were removed from the model and estradiol-to-progesterone ratio, testosterone, and cortisol were retained. Since some prior research on jealousy has implicated estradiol (e.g., Cobey et al., 2011), we also ran an additional model with estradiol as the only predictor. This model did not show a significant within-subject effect of estradiol ($t = -0.26$, unstandardized beta = -0.039 , $p = .79$). Repeating these analyses controlling for the possible effects of participant age on relationship jealousy showed the same pattern of results in all cases, except that the effect of partnership status was now only marginally significant ($p = .050$). None of these analyses showed significant effects of participant age.

4. Discussion

Our analyses of women's reported intrasexual competitiveness showed that women reported greater intrasexual competitiveness in test sessions where measured salivary testosterone was high. This within-subject effect of testosterone on intrasexual competitiveness was independent of the possible effects of estradiol, progesterone, estradiol-to-progesterone ratio, or cortisol, none of which had any significant effects. That the effect of testosterone was marginally significant when controlling for the effects of test session order ($p = .058$) also suggests that the observed effect of testosterone on reported intrasexual competitiveness was unlikely to be simply an artifact of order effects. These results complement Cobey et al.'s (2013) suggestion that reported intrasexual competitiveness varies as a function of women's testosterone level. By contrast with Cobey et al. (2013), who inferred possible hormonal mechanisms by considering how hormonal contraceptive use affects hormone levels in women but did not measure women's actual hormone levels, our study is the first to link within-subject changes in reported intrasexual competitiveness directly to natural variation in women's measured testosterone levels.

As well as supporting Cobey et al.'s (2013) speculations about the hormonal mechanisms for within-subject changes in women's intrasexual competitiveness, our results for women's

testosterone levels also complement recent work in which priming women's intrasexual competitiveness increased their testosterone levels (Maner & McNulty, 2013; Ritchie & van Anders, 2014). These findings raise the possibility that our results for testosterone and intrasexual competitiveness simply reflect women having experienced more intrasexual competitiveness prior to some test sessions than others (i.e., our results could reflect the effects of experiencing intrasexual competitiveness on endogenous testosterone levels, rather than the effects of endogenous testosterone levels on intrasexual competitiveness). However, the results of research in which *both* testosterone and cortisol levels were measured before and after intrasexual competition has found that engaging in intrasexual competition increases both women's testosterone and cortisol (Bateup et al., 2002; Casto & Edwards, in press). Thus, if our results simply reflected the effects of experiencing intrasexual competitiveness on endogenous hormone levels, we would have expected to see positive within-subject effects of both testosterone and cortisol levels on intrasexual competitiveness. By contrast with this prediction, we observed only an effect of testosterone in the current study, suggesting that our results are not simply due to the effects of experiencing intrasexual competition on hormone levels. Our proposal that our results are more likely to reflect the effects of testosterone on intrasexual competitiveness (rather than vice versa) is also supported by Cobey et al.'s (2013) results indicating that partnered women's intrasexual competitiveness decreased once they started using hormonal contraceptives, which would lower their testosterone levels (Zimmerman et al., 2014). It is also consistent with research reporting that exogenous testosterone increases intrasexual competitiveness in female birds and rats (e.g., Albert et al., 1990; Zysling et al., 2006). Nonetheless, we acknowledge here that additional work in which women's testosterone is experimentally manipulated is needed to further clarify the causal direction of the relationship between within-subject changes in testosterone and intrasexual competitiveness. We also acknowledge that additional work investigating the psychological mechanisms through which changes in testosterone influence intrasexual competitiveness

(e.g., the possible role of changes in sexual desire) would be required to have a full understanding of the mechanisms that regulate women's intrasexual competitiveness.

While Cobey et al. (2013) found that intrasexual competitiveness was decreased following hormonal contraceptive use in a sample of partnered women (N=14), they observed no effect of hormonal contraceptive use on unpartnered women's intrasexual competitiveness (N=14). By contrast, we observed no interaction between the effects of women's testosterone level and partnership status in our study of 46 partnered and 90 unpartnered women. Although the reasons for this apparent discrepancy are currently unclear, it is possible that the null result for unpartnered women in Cobey et al. (2013) is a false negative arising from the relatively small sample size.

Although our analyses suggest that reported intrasexual competitiveness tracks changes in women's testosterone levels, the functions (if any) of testosterone-linked changes in intrasexual competitiveness remain unclear. Although there are several reasons to think that our findings for testosterone and intrasexual competitiveness are unlikely to simply be a consequence of the effects of intrasexual competitiveness on testosterone levels (see earlier discussion), one possibility is that the tendency for intrasexual competitiveness to track changes in women's testosterone level across the five test sessions is a low-cost functionless byproduct of responses that function primarily to increase intrasexual competitiveness in situations where competition for resources triggers a facultative increase in testosterone (Maner & McNulty, 2013; Ritchie & van Anders, 2014). Alternatively, it could be a low-cost functionless byproduct of processes that function primarily to increase intrasexual competitiveness in response to changes in testosterone that occur over longer timespans, such as over pubertal development. Since some (but not all) studies have reported small increases in testosterone around ovulation (Caruso et al., 2014; Dabbs, 1990; Dabbs & de La Rue, 1991), we do not discount the possibility that increased intrasexual competitiveness when testosterone is high may occur because of direct or indirect benefits

associated with increased intrasexual competitiveness around ovulation (see Fisher, 2004). However, on this matter, it is important to note that Cobey et al. (2013) observed no difference in reported intrasexual competitiveness between the late-follicular and mid-luteal phases of the menstrual cycle in women with natural menstrual cycles and that we found no evidence that women's intrasexual competitiveness tracked changes in their estradiol-to-progesterone ratio, a correlate of women's fertility (Baird et al., 1991; Landgren et al., 1980). Further work is needed to explore these issues. Further work is also needed to establish whether the relationship between changes in testosterone and intrasexual competitiveness occurs in women whose hormonal profiles have been altered by factors known to impact endogenous hormones, such as obesity, smoking, and polycystic ovarian syndrome.

Although the within-subject effects of testosterone on reported intrasexual competitiveness were significant when testosterone was included as the only predictor ($p=.013$) and when controlling for other potential hormonal predictors ($p=.041$) and was marginally significant when controlling for effects of test session order ($p=.058$), the effects were generally weak. Thus, although we note here that there is converging evidence that reported intrasexual competitiveness tracks changes in testosterone from previous work that used the same intrasexual competitiveness scale to show an effect of oral contraceptive use on intrasexual competitiveness (Cobey et al., 2013) and work showing that exogenous testosterone increases intrasexual competitiveness in some non-human animals (e.g., Albert et al., 1990; Zysling et al., 2006), further research is needed to establish how robust the link between testosterone and intrasexual competitiveness that was observed in the current study actually is. Further work is also needed to establish whether the effect of testosterone on intrasexual competitiveness in women is as weak as is suggested by our current results or whether the weak results are a consequence of using a questionnaire to assess intrasexual competitiveness and/or the non-competitive laboratory setting in which the study was conducted.

While our analyses of intrasexual competitiveness revealed a significant positive effect of testosterone, we found little evidence that jealousy tracked changes in women's hormone levels. In particular, none of our analyses revealed the positive effects of estradiol that would have been predicted by previous work linking jealousy to estrogen dose in hormonal contraceptives (Cobey et al., 2011) or differences among women in their salivary estradiol (Geary et al., 2001). Importantly, that testosterone had a positive effect on women's reported intrasexual competitiveness, but not reported jealousy, indicates that the effect of testosterone on reported intrasexual competitiveness is not simply an artifact of a possible general response bias, whereby testosterone may have made women more willing to use extreme points on response scales.

In conclusion, our analyses of reported intrasexual competitiveness suggest that intrasexual competitiveness tracks within-subject changes in women's testosterone, but not estradiol, progesterone, estradiol-to-progesterone ratio, or cortisol. By contrast with this finding for intrasexual competitiveness and testosterone, we found no evidence that relationship jealousy tracked changes in women's hormone levels. These results support previous speculation that testosterone plays a key role in regulating women's intrasexual competitiveness (Cobey et al., 2013), but calls into question the suggested role of estradiol in women's relationship jealousy (Cobey et al., 2011; Geary et al., 2001). While previous research on changes in women's intrasexual competitiveness has highlighted links with estimated fertility (Durante et al., 2008, 2011; Fisher, 2004; Haselton et al., 2007; Lucas & Koff, 2013; Vukovic et al., 2009), our study is the first to demonstrate correlated changes in intrasexual competitiveness and natural variation in women's testosterone levels.

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7. Figures

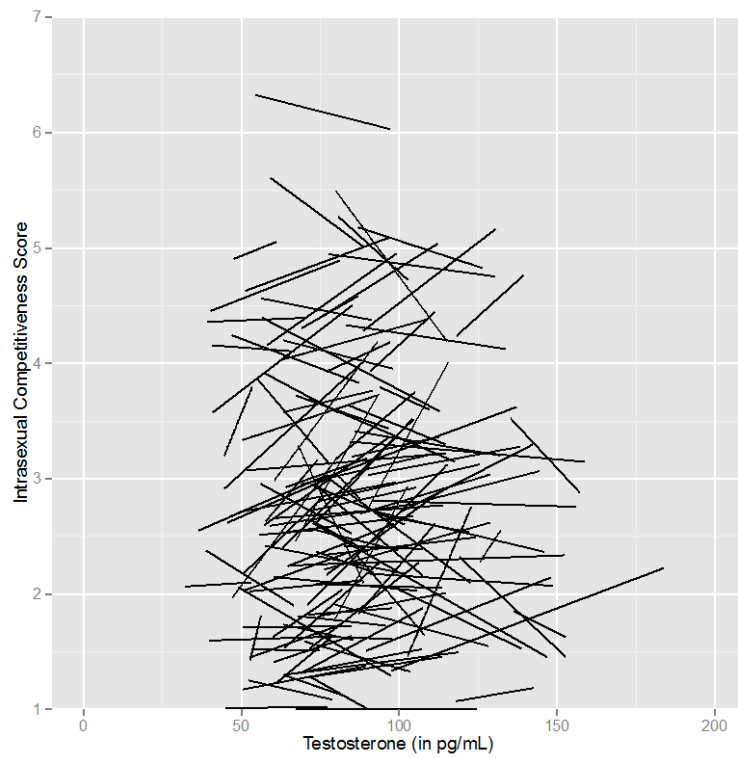


Figure 1. Change in reported intrasexual competitiveness as a function of testosterone.

Graphs show lines of best fit for individual participants.